



The MtSNF4b subunit of the sucrose non-fermenting-related kinase complex connects after-ripening and constitutive defense responses in seeds of *Medicago truncatula*

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Mots-clés	biotic stress [7], dormancy [8], <i>Medicago truncatula</i> [9], seeds [10], SNF4b [11] Dormant seeds are capable of remaining alive in the hydrated state for extended periods of time without losing vigor, until environmental cues or after-ripening result in the release of dormancy. Here, we investigated the possible role of the regulatory subunit of the sucrose non-fermenting-related kinase complex, MtSNF4b, in dormancy of <i>Medicago truncatula</i> seeds. Expression of MtSNF4b and its involvement in a high-molecular-weight complex are found in dormant seeds, whereas imbibition of fully after-ripened, non-dormant seeds leads to dissociation of the complex. MtSNF4b is capable of complementing the yeast Δ snf4 mutant and of interacting with the MtSnRK1 α -subunit in a double hybrid system. Transcriptome analyses on freshly harvested and after-ripened RNAi Mtsnf4b and wild-type embryos implicate MtSNF4b in the defense response in hydrated dormant embryonic tissues, affecting the expression of genes encoding enzymes of flavonoid and phenylpropanoid metabolism, WRKY transcription factors and pathogenesis-related proteins. Silencing MtSNF4b also increased the speed of after-ripening during dry storage, an effect that appears to be related to a change in base water potential. No significant difference in ABA content or sensitivity was detected between mutant and wild-type seeds. Pharmacological studies using hexoses and sugar analogs revealed that mannose restored germination behavior and expression of the genes PAL, CHR and IFR in RNAi Mtsnf4b seeds towards that of the wild-type, suggesting that MtSNF4b might act upstream of sugar-sensing pathways. Overall, the results suggest that MtSNF4b participates in regulation of a constitutively activated defense response in hydrated, dormant seeds.
Résumé en anglais	

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